Changes in the Disparities in Chronic Diseases During the Course of the 20th Century

Robert W. Fogel

ABSTRACT Longitudinal studies support the proposition that the extent and severity of chronic conditions in middle and late ages are to a large extent the outcome of environmental insults at early ages, including in utero. Data from the Early Indicators Project, undertaken at the Center for Population Economics, suggest that the range of differences in exposure to disease has narrowed greatly over the course of the 20th century, that age-specific prevalence rates of chronic diseases were much lower at the end of the 20th century than they were at the beginning of the last century or during the last half of the 19th century, and that there has been a significant delay in the onset of chronic diseases over the course of the 20th century. These trends appear to be related to changes in levels of environmental hazards and in body size. These findings have led investigators to posit a synergism between technological and physiological improvements. This synergism has contributed to reductions in inequality in real income, body size, and life expectancy during the 20th century.

Graduate School of Business, University of Chicago, and the National Bureau of Economic Research, Cambridge, MA.

Correspondence: Graduate School of Business, University of Chicago, 5807 S. Woodlawn, Suite 367, Chicago, IL 60637.

E-mail: rwf@cpe.uchicago.edu.

T HE MAIN PROPOSITION OF THIS ARTICLE is that the extent and severity of chronic conditions in middle and late ages are to a large extent the outcome of environmental insults at early ages, including in utero. Many of these assaults are infectious diseases that occur at young ages. Evidence to support this proposition comes partly from the study being conducted at the University of Chicago and elsewhere on the aging of the Union Army Veterans, the Early Indicators Project. But it is also supported by other longitudinal studies in the United States and abroad.

Some recent studies indicate two-generation effects among humans. Perhaps the most widely cited case involves women who were born or were in gestation during the Dutch famine, which lasted about eight or nine months toward the end World War II and was precipitated by sharp reductions in rations. Although this famine resulted in an elevated perinatal death rate, it was for a long time thought that women who survived and were subsequently well fed were just as healthy as women who were born before the famine or afterwards. However, when these women themselves began giving birth, their children had elevated perinatal death rates. Of course, we will still have to wait to see what the effects of the famine will be on the rate of increase in chronic conditions and on longevity at older ages. However, there is evidence that children of mothers who were in the first trimester of gestation when the famine struck and who survived infancy have elevated prevalence of coronary heart disease now that they are in their mid-50s (Roseboom et al. 2000).

Environmental conditions were far more severe in 1900 than in recent decades. There are many ways this can be measured, one of which is the infant death rate. Figure 1 presents some results of an ongoing study of changes in disparities in infant mortality during the 20th century by neighborhood, based on the 24 largest U.S. cities in 1900. At the present time, we are using wards averaging about 30,000 people as the unit of observation. But such large units tend to mask disparities, since they tend to combine both rich and poor neighborhoods. We hope eventually to conduct the analyses at the level of census districts, which average about 8,000 individuals and are more homogenous with respect to socioeconomic status and health conditions than wards.

However, even when the analysis is conducted at the ward level, the results are striking. In Figure 1, the y axis is the infant death rate, and the x axis is year. There are three sets of observations: the maximum infant death rate across about 120 wards of six large cities are clustered around the top line; the minimum infant death rates are clustered around the bottom line; and the line in the middle represents the average. The main point of this figure is how wide the range in infant death rates were at the beginning of the period: 226 per thousand between the best and the worst wards in 1900. By 1950, the differential between the best and the worst wards had declined to about 38 per thousand. During the first half of the 20th century, the spread between the best and the worst wards diminished by 83%. Hence, the first point to be noted about changes and dis-
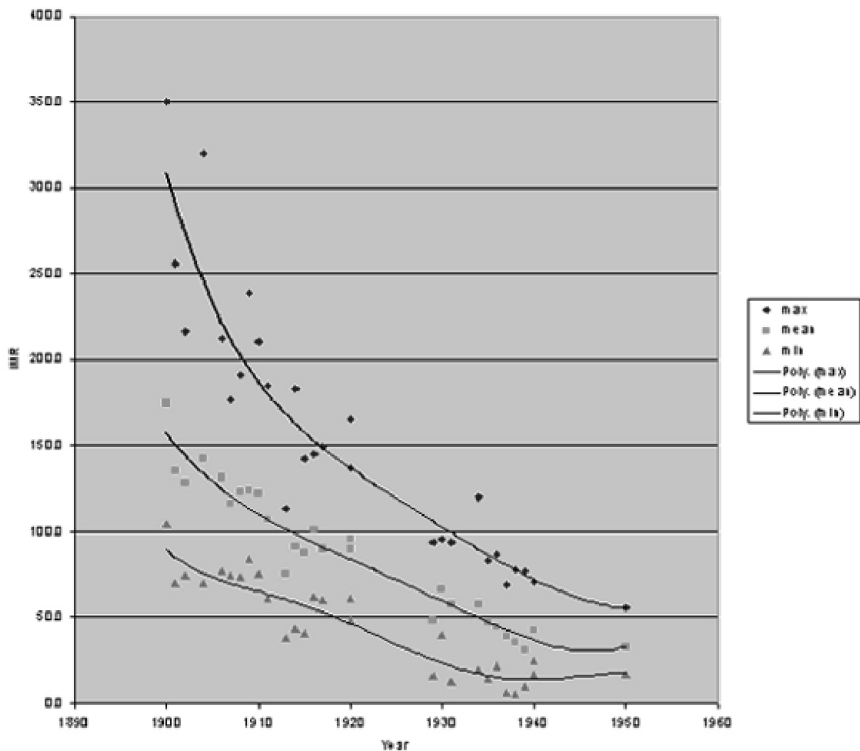
FIGURE 1

Preliminary findings on trends in disparities in infant death rates of the wards of six large U.S. cities, 1900–1950. This diagram shows that between 1900 and 1950 more than 80% of the difference in the infant mortality rate between the worst and best wards of six large cities had disappeared.

parities in the 20th century is that the range of differences in exposure to disease narrowed greatly over the course of the century. The disparities we observe today are much smaller than they used to be. From the perspective of 1900, we are currently engaged in fine-tuning, not in gross corrections of disparities.

Many examples of how much wider disparities were among ethnic groups can be presented. One example is from a working paper by Todd Bridges (2002). The infant death rate from diarrheal diseases during the summer months in Chicago in 1910 for foreign-born mothers was 9 times as high as the rate for native-born mothers (Bridges 2002).

I began this article by focusing on infectious diseases because one of the principal findings of the Early Indicators Project is that insults from infectious diseases at early ages have a large impact on the prevalence rates of chronic diseases and disabilities in middle and late ages. Another major finding of the project is that the age-specific prevalence rates of chronic diseases were much lower at the end of the 20th century than they were at the beginning of that century or dur-

TABLE 1 THE SHARE OF NORTHERN WHITE MALES OF MILITARY AGE UNFIT FOR MILITARY SERVICE IN 1861

Age	Cohort examined (%)	Examinees who were rejected (%)
16–19	80.9	16.0
20–24	70.4	24.5
25–29	52.3	35.8
30–34	41.0	42.9
35–39	41.6	52.9

ing the last half of the 19th century. The Union Army data reveal the ubiquity of chronic health conditions during the century before World War II. Not only was the overall prevalence rate of these diseases much higher among the elderly than today, but they afflicted the teens, young adults and middle ages to a much greater extent than today. This fact is brought out by Table 1, which shows that more than 80% of all males age 16 to 19 in 1861 and more than 70% of men age 20 to 24 were examined for the Union Army. These examinees were over-whelmingly volunteers (less than 4% were drafted), who presumably thought they were fit enough to serve. Yet disability rates were higher than today. Even among teenagers more than one of six was disabled, and among men age 35 to 39, more than half were disabled. Despite their relatively young ages, cardiovas-cular diseases (mainly rheumatic) accounted for 11% of the rejections; hernias another 12%; eye, ear, and nose diseases 7%; tuberculosis and other respiratory diseases 7%; and tooth and gum diseases 8%. Most of the other rejections were due to orthopedic conditions and general debility (Lee 2001).

These findings about the early onset of chronic diseases cast new light on the debate about the effect of increased longevity on the prevalence rates of chronic diseases. Those who argued that the effect of increased longevity was to increase the average duration of chronic disease assumed no delay in the average age of onset of these diseases. They were also influenced by cross-sectional evidence that showed some increases in disability rates during the 1970s and 1980s, despite the continuing decline in mortality rates (Riley 1990a, 1990b, 1991; Wolfe and Haveman 1990). It seemed plausible that various health interventions and envi-ronmental changes served to reduce the severity of diseases and thus delayed death without providing cures, as has been the case with AIDS.

However, as Table 2 shows, there has been a significant delay in the onset of chronic diseases during the course of the 20th century. Men age 50–54 were 24% more likely to be free of chronic conditions in 1994 than a century earlier. At age 60–64, white males today are 2.5 times more likely to be free of chronic diseases than their counterparts a century ago. Further light is shed on the issue

TABLE 2 INCREASE IN PROPORTION OF WHITE MALES
WITHOUT CHRONIC CONDITIONS DURING THE
20TH CENTURY

| Age | PROPORTION WITHOUT CHRONIC CONDITIONS | |
	1890–1910	circa 1994
50–54	0.33	0.41
55–59	0.21	0.29
60–64	0.10	0.25
65–69	0.03	0.14

TABLE 3 AVERAGE AGE OF ONSET OF CHRONIC CONDITIONS
AMONG AMERICAN MALES NEAR BEGINNING AND
END OF THE 20TH CENTURY

| Condition | AVERAGE AGE OF ONSET (YEARS) | |
	Men born 1830–1845	Men born 1918–1927
Heart disease	55.9	65.4
Arthritis	53.7	64.7
Neoplasm	59.0	66.6
Respiratory	53.8	65.0

by considering specific diseases (see Table 3). Arthritis began 11 years later
among men who turned 65 between 1983 and 1992 than among those who
turned age 65 between 1895 and 1910. The delay in the onset of a chronic con-
dition was about 9 years for heart diseases, about 11 years for respiratory diseases
(despite much higher rates of cigarette smoking), and nearly 8 years for neo-
plasms.[1]

Union Army veterans who endured poor health did not typically die quickly.
Veterans who lived to be at least age 50, and who entered the pension system
before age 51, lived an average of 24 years past age 50. Moreover, at their last
examination on or before age 51, their average degree of disability was 58%,
where 100% indicates complete incapacity for manual labor. Between age 50 and
60, disability ratings (controlled for age at death) continued to rise sharply, and
then increased at a decreasing rate. It is worth noting that of the veterans who
lived to be age 50, about 29% lived to be age 80 or more. For these "Old Old,"
the level of disability for manual labor averaged between 85 and 100% for a
decade or more. Indeed, some survived with such high levels of disability for as

---

[1]Since current techniques make it possible to diagnose heart disease and neoplasms sooner in the
development of these diseases than used to be the case c. 1910, the figures given in the text should
be considered lower bounds on the delay in the onset of these conditions.

TABLE 4   AVERAGE NUMBER OF COMORBIDITIES AMONG
            VETERANS WHO LIVED TO BE AT LEAST AGE 50

Average age of death	Veterans who lived to at least age 50, who died in interval (%)	Average number of comorbidities at last examination before death
50–54	3.9	4
55–59	6.4	5
60–64	9.8	6
65–69	14.0	6
70–74	18.3	7
75–79	19.1	7
80–84	15.5	7
85–89	9.0	8
90–94	3.4	8
95 or over	0.7	7

much as a quarter of a century (Helmchen 2003). As Table 4 shows, survivors usually acquired more and more comorbidities (the coexistence of two or more disease processes) as they aged. Those who lived past age 85 had twice as many co-morbidities as those who died by age 55.

Consideration of the sweep of the 20th century puts the debate over the relationship between the increase in life expectancy and the change in the burden of chronic disease among the elderly in a new perspective. It now appears that the decline in morbidity rates paralleled the decline in mortality rates. Indeed, the delay in the onset of chronic disabilities between 1900 and the 1990s for those who lived to age 50 was greater than the increase in life expectancy at age 50 over the same periods. The average delay in the onset of chronic conditions over the century was more than 10 years (Helmchen 2003).[2] However, the average increase in male life expectancy was about 6.6 years (Bell, Wade, and Goss 1992).

Public health policies before 1940 had a large impact on the decline in chronic disability rates decades later. Dora Costa (2000) has estimated the impact of public health and socioeconomic status factors at late developmental and young adult ages on risks of incurring chronic conditions at middle and late ages. Significant predictors included mortality rates in counties of enlistments, infectious diseases experienced during the Civil War, and being a prisoner of war. She

---

[2]The delay in the average age of onset in chronic diseases can be decomposed into two parts: (1) the shift in the age-specific disease schedule; and (2) the change in the distribution of ages due to the increase in life expectancy and the decline in the fertility rate. We have not yet completed this decomposition. However, preliminary estimates indicate that the contribution of the change in the age distribution was small.

focused on a set of chronic conditions for which clinical diagnoses were essentially the same in the early 1900s as today (such as lower back pain, joint problems, decreased breath or adventitious sounds, irregular pulse, and valvular heart disease). This procedure permitted her to estimate how much of the observed decline in the prevalence rates of comparable conditions was due to the reduction in specific risk factors. Prevalence rates for 1971–1980 were computed from the National Health and Nutrition Examination Survey (NHANES).

Costa found that elimination of exposure to specific infectious diseases during developmental and young adult ages explained between 10 and 25% of the declines in the specified chronic diseases of middle and late ages between 1900–1910 and 1971–1980. Occupational shifts were also important, accounting for 15% of the decline in joint problems, 75% of the decline in back pains, and 25% of the decline in respiratory diseases.

Costa (2002) pushed this line of analysis further by documenting the decline in functional limitations among U.S. men between ages 50 and 74 over the course of the 20th century. A central issue is the factoring of the decline in functional limitations among three processes: the decline in the prevalence rates of specific chronic diseases, the reduction in the debilitating sequelae of these diseases, and the influence of new medical technologies that relieve and control the sequelae. Her analysis turned on five functional limitations: difficulty walking, difficulty bending, paralysis, blindness in at least one eye, and deafness in at least one ear. Prevalence rates of these limitations among men aged 50 to 74 were computed for the Union Army, NHANES (1988–1994), and NHIS (1988–1994).

On average, these five functional limitations declined by about 40% during the course of the 20th century. Using probit regressions, Costa attributed 24% of the decline to reduction in the debilitating effect of chronic conditions, and 37% to the reduced rates of chronic conditions.

The Significance of Changes in Body Size

The contribution of improvements in body size as measured by stature, BMI, and other dimensions have run through the research of the Early Indicators Project like a red line. The discovery of correlations in time series going back to the colonial period between changes in stature and changes in life expectancy for the United States were reported first in 1986, although it was known as early as 1978.[3] Pursuit of a variety of issues called attention to the significance of changes in body size to the long-term decline in chronic conditions and mortality. For example, Diane Lauderdale and Paul Rathouz (1999) sought to inves-

---

[3]For reviews of earlier work dealing with the use of height, BMI and other anthropometric measures as indexes of changes in health and the standard of living over time, see Komlos and Cuff 1998; Steckel 1995.

tigate the impact of unhealthy environments on the genetic component of height. They hypothesized that an unhealthy environment might attenuate the effects of genotype, and to test that hypothesis, they constructed a sample of brothers who served in the Union Army. Their analysis showed that brothers from unhealthy counties had both higher variances in height and lower covariance in the heights of siblings than was expected from standard equations for measuring genetic influences in the heights of siblings. Study of the likelihood of developing specific diseases while in the service also pointed to the importance of stature. For example, short recruits were more likely to develop tuberculosis while in service than taller ones (Birchenall 2003; see also Lee 1997).

In 1995, Dora Costa discovered a sample of 23,000 Union Army recruits who were, for scientific reasons, more intensively examined than the typical recruit (Costa n.d.). Benjamin A. Gould, a leading astronomer and one of the founders of the National Academy of Sciences, who was in charge of the project, collected information on waist and hip circumference, lifting strength, vital capacity of lungs, height, weight, shoulder breadth, and chest circumference. The sample covered whites, blacks, and Native Americans. Costa linked a subsample of 521 white recruits who survived to 1900 to their pension records. She also compared the Union Army soldiers with soldiers measured in 1946–1947, 1950, and 1988. Over a span of 100 years, men in the military became taller and heavier. Their height increased by 5cm, and the BMIs of men age 31 to 35 increased from 23 to 26. Controlling for BMI and age, the waist–hip and chest–shoulder ratios (both measures of abdominal fat) were significantly greater in the Gould sample than in the 1950 and 1988 samples.

Using an independent competing risk hazard model to estimate the effect of changes in body shape on the risk of death from cerebrovascular and ischemic heart disease at older ages, Costa found that a low waist-to-hip ratio increased mortality by 4.4 times relative to the mean and controlling for BMI, while a high waist–hip ratio increased mortality risk by 2.9 times. Substituting the characteristics of soldiers in 1950 (who reached age 65 or over during the late 1980s) into her regression model produced a 15% decline in all-cause mortality above age 64, implying that changes in frame size explain about 47% of the total decline in all-cause mortality at older ages between the beginning and the end of the 20th century.

The Theory of Technophysio Evolution

Recognition of environmentally induced changes in human physiology during the 20th century that had a profound impact on the process of aging did not become apparent to Early Indicators Project investigators until mid-1993. The key finding was that prevalence rates in the main chronic diseases among Union Army veterans age 65 and older were much higher in 1910 than among veterans of World War II of the same ages during the mid-to-late 1980s. That finding

was first set forth in a 1993 working paper and was elaborated and subsequently characterized as a "theory of technophysio evolution." The theory of techno-physio evolution arose out of intense discussion among the senior investigators, consultants, and research assistants during 1993–1994, with the physicians providing much of the intellectual leadership, especially Nevin Scrimshaw, J. M. Tanner, and Irving Rosenberg (see Fogel n.d.). This theory points to the existence of a synergism between technological and physiological improvements that has produced a form of human evolution that is biological but not genetic, rapid, culturally transmitted, and not necessarily stable. The process is still ongoing in both rich and developing countries.

Unlike the genetic theory of evolution through natural selection, which applies to the whole history of life on earth, technophysio evolution applies only to the last 300 years of human history, and particularly to the last century.[4] The theory of technophysio evolution rests on the proposition that during the last 300 years, particularly during the last century, human beings have gained an unprecedented degree of control over their environment—a degree of control so great that is sets them apart not only from all other species, but also from all previous generations of Homo sapiens. This new degree of control has enabled Homo sapiens to increase its average body size by over 50%, to increase its average longevity by more than 100%, and to greatly improve the robustness and capacity of vital organ systems.[5]

Figure 2 helps to point up how dramatic the change in the control of the environment after 1700 has been. During its first 200,000 or so years, Homo sapiens increased at an exceedingly slow rate. The discovery of agriculture about 11,000 years ago broke the tight constraint on the food supply imposed by a hunting and gathering technology, making it possible to release between 10 and 20% of the labor force from the direct production of food and also giving rise to the first cities. The new technology of food production was so superior to the old one that it was possible to support a much higher rate of population increase than had existed prior to c. 9000 BCE. Yet, as Figure 2 shows, the advances in the technology of food production after the *second* Agricultural Revolution (which began about AD 1700) were far more dramatic than the earlier breakthrough, since they permitted the population to increase at so high a rate that the line of population appears to explode, rising almost vertically. The new technological breakthroughs in manufacturing, transportation, trade, communications, energy

---

[4]Fogel and Costa (1997) limit technophysio evolution to the last 300 years for two reasons. It was not until about 1700 that changes in technology permitted population growth far in excess of previous rates. Moreover, after 1700 body weight and stature increase to unprecedented levels.

[5]Although a considerable body of empirical evidence has accumulated indicating that a "good" environment both speeds up biological development at young ages and delays the onset of chronic conditions at middle and late ages, there is as yet no agreed-upon theory about the cellular and molecular processes that explains these observations.
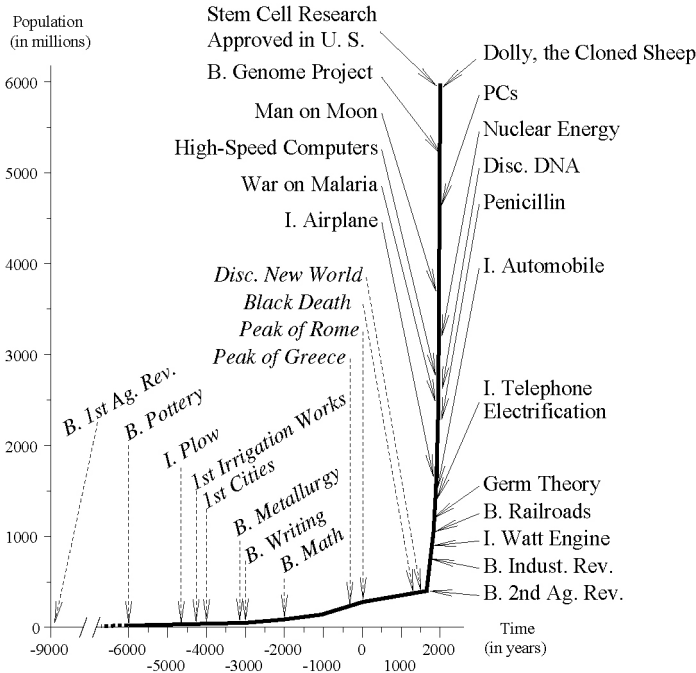
Population
(in millions)

Stem Cell Research
Approved in U. S.

Dolly, the Cloned Sheep

B. Genome Project

PCs

Man on Moon

Nuclear Energy

High-Speed Computers

Disc. DNA

War on Malaria

Penicillin

I. Airplane

I. Automobile

Disc. New World
Black Death
Peak of Rome
Peak of Greece

I. Telephone
Electrification

B. 1st Ag. Rev.

B. Pottery

I. Plow

1st Irrigation Works

1st Cities

B. Metallurgy

B. Writing

B. Math

Germ Theory

B. Railroads

I. Watt Engine

B. Indust. Rev.

B. 2nd Ag. Rev.

Time
(in years)

-9000   -6000   -5000   -4000   -3000   -2000   -1000   0   1000   2000

6000
5000
4000
3000
2000
1000
0

Figure 2

The growth of world population and some major events in the history of technology. There is usually a lag between the invention (I) of a process or a machine and its general application to production. "Beginning" (B) usually means the earliest stage of this diffusion process.

production, leisure-time services, and medical services were in many respects even more striking than those in agriculture. Figure 2 emphasizes the huge acceleration in both population and technological change during the 20th century. The increase in the world's population between 1900 and 1990 was four times as great as the increase during the whole previous history of humankind.

Technophysio Evolution and the Reduction in Inequality During the 20th Century

The 20th century contrasts sharply with the record of the two preceding centuries. In every measure that we have bearing on the standard of living, such as real income, homelessness, life expectancy, and height, the gains of the lower classes have been far greater than those experienced by the population as a whole, whose overall standard of living has also improved.

The "Gini ratio," which is also called the "concentration ratio," is the measure of the inequality of the income distribution most widely used by econo-

mists.[6] This measure varies between 0 (perfect equality) and 1 (maximum inequality). In the case of England, for example, which has the longest series of income distributions, the Gini ratio stood at about 0.65 near the beginning of the 18th century, at about 0.55 near the beginning of the 20th century, and at 0.32 in 1973, when it bottomed out, not only in Britain, but also in the United States and other rich nations.[7] This measure indicates that over two-thirds of the reduction in the inequality of income distributions between 1700 and 1973 took place during the 20th century. The large decrease in such inequality, coupled with the rapid increase in the average real income of the English population, means that the per capita income of the lower classes was rising much more rapidly than those of the middle or upper classes.[8]

A similar conclusion is implied by the data on life expectancies. For the cohort born about 1875, there was a gap of 17 years between the average length of life of the British elite and of the population as a whole. There is still a social gap in life expectancies among the British, but today the advantage of the richest classes over the rest of the population is only about four years. Thus about three-quarters of the social gap in longevity has disappeared. As a consequence, the life expectancy of the lower classes increased from 41 years at birth in 1875 to about 74 years today, while the life expectancy of the elite increased from 58 years at birth to about 78 years. If anything sets the 20th century apart from the past, it is this huge increase in the longevity of the lower classes.[9]

Data on stature also indicate the high degree of inequality during the 19th century. At the close of the Napoleonic wars, a typical British male worker at maturity was about five inches shorter than a mature male of upper class birth. There is still a gap in stature between the workers and the elite of Britain, but now the gap is only on the order of an inch. Height differentials by social class have virtually disappeared in Sweden and Norway but not yet in the United States. Statistical analysis across a wide array of rich and poor countries today shows a strong correlation between stature and the Gini ratio (Rona, Swan, and Altman 1978; Steckel 1995).

Weight is another important measure of inequality. Despite the great emphasis in recent years on weight reduction, the world still suffers more from undernutrition and underweight than from overweight, as the World Health Organiza-

---

[6]Stature and Gini ratios are significantly correlated, but as the following discussion of height and BMI indicates, the anthropometric measures reveal important aspects of welfare that are not as apparent in the movement of Gini ratios.

[7]There has been a rise in the Gini ratio since 1973 in virtually all the rich nations for which such information is available (see Fogel 2000).

[8]On trends in the Gini ratio between c. 1690 and 1973 and the debate over this trend, see Feinstein 1988; Floud et al. 1990; Fogel 2000; Lindert and Williamson 1982, 1983; Soltow 1968; Williamson 1985.

[9]Data for males are presented in Case et al. 1962; Hattersley 1999; Hollingsworth 1977.

tion has repeatedly pointed out. Although one should not minimize the afflictions caused by overnutrition, it is important to recognize that even in rich countries such as the United States, undernutrition remains a significant problem, especially among impoverished pregnant women, children, and the aged.

The secular increase in body builds is due primarily to the great improvement in socioeconomic conditions over the past several centuries, rather than to genetic factors, as can be seen by considering Holland. The average height of young adult males was only 64 inches in that country during the middle of the 19th century. The corresponding figure today is about 72 inches. An increase of eight inches in just four generations cannot be due to natural selection or genetic drift, because such processes require much longer time spans. Nor can it be attributed to heterosis (hybrid vigor), because Holland's population has remained relatively homogeneous and because the effects of heterosis in human populations have been shown both empirically and theoretically to have been quite small. It is hard to come up with credible explanations for the rapid increase in heights that do not turn on environmental factors, especially improvements in nutrition and health. These environmental factors appear to be still at work. Stature is still increasing, although at a somewhat slower rate, and nations have not yet reached a mean height that represents the biological limit of humankind under current biomedical technology (Drukker 1994; Drukker and Tassenaar 1997; Fogel 1992; Schmidt, Jorgenson, and Michaelsen 1995; van Wieringen 1986).

Homelessness is another indicator of the dramatic reduction in inequality during the 20th century. Down to the middle of the 19th century, between 10 and 20% of the population in Britain and on the Continent were homeless persons whom officials classified as vagrants and paupers. Estimates of vagrancy and pauper rates in the United States during the 19th century are less certain, but these rates appear to have reached European levels in the major cities during the middle decades of that century. When we speak of homelessness in the United States today, we are talking about rates below 0.4% of the population. Many of the homeless today are mentally ill individuals prematurely released from psychiatric institutions that are inadequately funded. Many others are chronically poor and inadequately trained for the current job market (Cipolla 1980; Colquhoun 1814; Fogel 1987, 1989, 1993; Hannon 1984a, 1984b, 1985; Himmelfarb 1983; Jencks 1994; Laslett 1965; Lindert and Williamson 1982; Soltow 1968).

The relatively generous poverty program developed in Britain during the second half of the 18th century, and the bitter attacks on that program by Malthus and others, have given the unwarranted impression that government transfers played a major role in the secular decline in beggary and homelessness. Despite the relative generosity of English poor relief between 1750 and 1834, beggary and homelessness fluctuated between 10 to 20%. Despite the substantial reduction in the proportion of national income transferred to the poor as a result of the poor laws of 1834 and later years, homelessness declined sharply during the late 19th and early 20th centuries.

The fact is that government transfers were incapable of solving the problems of beggary and homelessness during the 18th and much of the 19th centuries, because the root cause of the problems was chronic malnutrition. Even during the most generous phases of the relief program, the bottom fifth of the English population was so severely malnourished that it lacked the energy for adequate levels of work (Fogel 1997; Fogel, Floud, and Harris n.d.).

At the end of the 18th century British agriculture, even when supplemented by imports, was simply not productive enough to provide more than 80% of the potential labor force with enough calories to sustain regular manual labor. It was the huge increases in English productivity during the later part of the 19th and the early 20th centuries that made it possible to feed even the poor at relatively high caloric levels. Begging and homelessness were reduced to exceedingly low levels, by 19th-century standards, only when the bottom fifth of the population acquired enough calories to permit regular work. The principal way in which government policy contributed to that achievement was through its public health programs. By reducing exposure to disease, more of the calories that the poor ingested were made available for work.

Conclusion

Technophysio evolution implies that some theoretical propositions that underlie some current economic models are misspecified because the initial endowments of health capital of successive cohorts increased over the course of the 20th century. The common assumption that the endowment of human physiological capacity is fixed, so that medical intervention can only slow down the rate of deterioration in the original endowment, means that ways of forecasting future improvement in human physiology are sometimes neglected and possible paths of increase in health endowments play little role in forecasting future health care costs or longevity.[10]

The theory of technophysio evolution implies that health endowments in a given population change with the year of birth. It also points to complex interactions between date of birth and the outcome of exposures to given risk factors. Hence, not all improvements in the outcome of exposure to health risks between, say, 1970 and 1990 are due to health interventions during that period. Improvements in life expectancy may depend only partly on the more effective medical technologies of those years. It could also reflect the improved physiologies experienced by later birth cohorts that are due to improved technologies in food production, public health practices, personal hygiene, diets, and medical interventions put into place decades before 1970, and hence cannot be attributed exclusively, perhaps even primarily, to health inputs between 1970 and

---

[10]Among the exceptions are Dasgupta 1993; Rosenzweig and Schultz 1988.

1990.[11] Moreover, many health interventions which are effective at late ages today would not be feasible if the level of physiological capital at late ages was as low today as it was at the beginning of the 20th century.

References

Allen, R. C. 1992. *Enclosure and the yeoman: The agricultural development of the South Midlands, 1450–1850*. Oxford: Oxford Univ. Press.

Allen, R. C. 1994. Agriculture during the industrial revolution. In *The economic history of Britain since 1700. Vol. 1, 1700–1860*, 2nd ed., ed. R. Floud and D. McCloskey, 96–122. Cambridge: Cambridge Univ. Press.

Barker, D. J. P. 1998. *Mothers, babies and disease in later life*. Edinburgh: Churchill Livingstone.

Birchenall, J. A. 2003. Airborne diseases: Tuberculosis in the Union Army. Paper presented at the Joint Meeting of the Early Indicators of Later Work Levels, Disease, and Death Program Project and Cohort Studies groups, National Bureau of Economics, Cambridge, MA, April 25–26.

Bridges, T. A. 2002. Preliminary findings on the linkage between infant mortality and education during early 20th-century Chicago. Working paper, Center for Population Economics, Univ. of Chicago. http://www.cpe.uchicago.edu/publication/publication.html.

Case, R. A. M., et al. 1962. *Chester Beatty Research Institute abridged serial life tables, England and Wales 1841–1960, Part 1*. London: Chester Beatty Research Institute.

Cipolla, C. M. 1974. *The economic history of world population*. Harmondsworth: Penguin.

Cipolla, C. M. 1980. *Before the industrial revolution: European society and economy, 1000–1700*. New York: W. W. Norton.

Clark, G. 1961. *World prehistory: An outline*. Cambridge: Cambridge Univ. Press.

Colquhoun, P. 1814. *Treatise on the wealth, power, and resources of the British Empire*. London: Joseph Mawmay.

Costa, D. L. 2000. Understanding the 20th-century decline in chronic conditions among older men. *Demography* 37(1):53–72.

Costa, D. L. 2002. Changing chronic disease rates and long-term declines in functional limitation among older men. *Demography* 39(1):119–37.

Costa, D. L. n.d. The measure of man and older age mortality: Evidence from the Gould sample. *J Econ Hist*, forthcoming.

Dasgupta, P. 1993. *An inquiry into well-being and destitution*. Oxford: Clarendon.

Derry, T. K., and T. I. Williams. 1960. *A short history of technology*. London: Oxford Univ. Press.

Drukker, J. W. 1994. The tradition of anthropometric history in the Netherlands. Paper presented at the National Bureau of Economic Research, Cambridge, MA, July 11–12.

Drukker, J. W., and V. Tassenaar. 1997. Paradoxes of modernization and material well-

---

[11] Much recent research indicates that waiting time to the onset of chronic diseases is a function of exposure to insults in utero and infancy. See Barker 1998; Scrimshaw 1997.

being in the Netherlands during the 19th century. In *Health and welfare during industrialization*, ed. R. H. Steckel and R. Floud, 331–77. Chicago: Univ. of Chicago Press.

Fagan, B. M. 1977. *People of the earth*, 2nd ed. Boston: Little, Brown.

Feinstein, C. H. 1988. The rise and fall of the Williamson Curve. *J Econ Hist* 48:699–729.

Floud, R., et al. 1990. *Height, health, and history: Nutritional status in the United Kingdom, 1750–1980*. Cambridge: Cambridge Univ. Press.

Fogel, R. W. 1987. Biomedical approaches to the estimation and interpretation of secular trends in equity, morbidity, mortality, and labor productivity in Europe, 1750–1980. Typescript, Center for Population Economics, Univ. of Chicago.

Fogel, R. W. 1989. *Without consent or contract*, vol. 1. New York: Norton.

Fogel, R. W. 1992. Introduction: Notes on the art of empirical research in the social sciences during an age of plunging costs in data processing. In *Without consent or contract. Vol. 2, Evidence and Methods*, ed. R. W. Fogel, R. A. Galantine, and R. L. Manning, 1–41. New York: Norton.

Fogel, R. W. 1993. New sources and new techniques for the study of secular trends in nutritional status, health, mortality, and the process of aging. *Hist Methods* 26(1):5–43.

Fogel, R. W. 1997. Economic and social structure for an ageing population. *Philos Trans R Soc Lond B Biol Sci* 352(1363):1905–17.

Fogel, R. W. 2000. *The fourth great awakening and the future of egalitarianism*. Chicago: Univ. of Chicago Press.

Fogel, R. W. n.d. Changes in the process of aging during the 20th century: Findings and procedures of the Early Indicators Project. *Population Develop Rev*, forthcoming.

Fogel, R. W., and D. L. Costa. 1997. A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs. *Demography* 34(1):49–66.

Fogel, R. W., R. Floud, and B. Harris. n.d. *A treatise on technophysio evolution and consumption*.

Hannon, J. U. 1984a. The generosity of antebellum poor relief. *J Econ Hist* 44:810–21.

Hannon, J. U. 1984b. Poverty in the antebellum northeast: The view from New York State's poor relief rolls. *J Econ Hist* 44:1007–32.

Hannon, J. U. 1985. Poor relief policy in antebellum New York State: The rise and decline of the Poorhouse. *Explorations Econ Hist* 22:233–56.

Hattersley, L. 1999. Trends in life expectancy by social class: An update. *Health Stat Q* 2:16–24.

Helmchen, L. 2003. Changes in the pattern of chronic disease among elderly Americans, 1870–2000. Working paper, Center for Population Economics, Univ. of Chicago. http://www.cpe.uchicago.edu.

Himmelfarb, G. 1983. *The idea of poverty: England in the early industrial age*. New York: Random House.

Hollingsworth, T. H. 1977. Mortality in the British peerage families since 1600. *Population* 32 (numéro special):323–52.

Jencks, C. 1994. *The homeless*. Cambridge: Harvard Univ. Press.

Komlos, J., and T. Cuff, eds. 1998. *Classics in anthropometric history*. St. Katharinen, Ger.: Scripta Mercaturae.

Laslett, P. 1965. *The world we have lost: England before the industrial age*. Rpt. New York: Scribner's, 1984.

Lauderdale, D. S., and P. J. Rathouz. 1999. Evidence of environmental suppression of familial resemblance: Height among U.S. Civil War brothers. *Ann Hum Biol* 26(5):413–26.

Lee, C. 1997. Socioeconomic background, disease, and mortality among Union Army recruits: Implications for economic and demographic history. *Explorations Econ Hist* 34(1):27–55.

Lee, C. 2001. Exposure to disease during growing ages and service. In *Early indicators of later work levels, disease, and death*, program project proposal submitted to the NIA. Typescript, Center for Population Economics, Univ. of Chicago.

Lindert, P. H., and J. G. Williamson. 1982. Revising England's social tables: 1688–1812. *Explorations Econ Hist* 19:385–408.

Lindert, P. H., and J. G. Williamson. 1983. English workers' living standards during the industrial revolution: A new look. *Econ Hist Rev* 36(1):1–25.

McNeill, W. 1971. *A world history*, 2nd ed. New York: Oxford Univ. Press.

Piggott, S. 1965. *Ancient Europe from the beginnings of agriculture to classical antiquity*. Chicago: Aldine.

Riley, J. C. 1990a. Long-term morbidity and mortality trends: Inverse health transitions. In *What we know about health transition: The cultural, social and behavioural determinants of health*. Proceedings of an International Workshop, Canberra, May 1989. Canberra: Health Transition Centre, Australian National University.

Riley, J. C. 1990b. The risk of being sick: Morbidity trends in four countries. *Population Develop Rev* 16(3):403–32.

Riley, J. C. 1991. The prevalence of chronic diseases during mortality increase: Hungary in the 1980s. *Population Stud* 45(3):489–96.

Rona, R. J., A. V. Swan, and D. G. Altman. 1978. Social factors and height of primary schoolchildren in England and Wales. *J Epidemiol Comm Health* 32:147–54.

Roseboom, T. J., et al. 2000. Coronary heart disease after prenatal exposure to the Dutch famine, 1944–45. *Heart* 84(6):595–98.

Rosenzweig, M. R., and T. P. Schultz. 1988. The stability of household production technology: A replication. *J Hum Resources* 23(4):535–49.

Schmidt, I. M., M. H. Jorgensen, and K. F. Michaelsen. 1995. Height of conscripts in Europe: Is postneonatal mortality a predictor? *Ann Hum Biol* 22:57–67.

Scrimshaw, N. S. 1997. More evidence that foetal nutrition contributes to chronic disease in later life. *BMJ* 315(7112):825–26.

Slicher van Bath, B. H. 1963. *The agrarian history of Western Europe, AD 500–1850*, trans. O. Ordish. London: Edward Arnold.

Soltow, L. 1968. Long-run changes in British income inequality. *Econ Hist Rev* 21:17–29.

Steckel, R. H. 1995. Stature and the standard of living, *J Econ Lit* 33(4):1904–40.

Trewartha, G. T. 1969. *A geography of populations: World patterns*. New York: John Wiley.

van Wieringen, J. C. 1986. Secular growth changes. In *Human growth: A comprehensive treatise, vol. 3, Methodology. Ecological, genetic, and nutritional effects on growth*, 2nd ed., eds. F. Falkner and J. M. Tanner, 307–71. New York: Plenum Press.

Williamson, J. G. 1985. *Did British capitalism breed inequality?* Boston: Allen and Unwin.

Wolfe, B. L., and R. Haveman. 1990. Trends in the prevalence of work disability from 1962 to 1984, and their correlates. *Milbank Q* 68(1):53–80.

Wrigley, E. A. 1969. *Population and history*. London: Weidenfeldt and Nicolson.